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ARCHIVES OF PEDIATRICS

A MONTHLY DEVOTED TO THE

DISEASES OF INFANTS AND CHILDREN

JOHN FITCH LANDON, M.D., Editor

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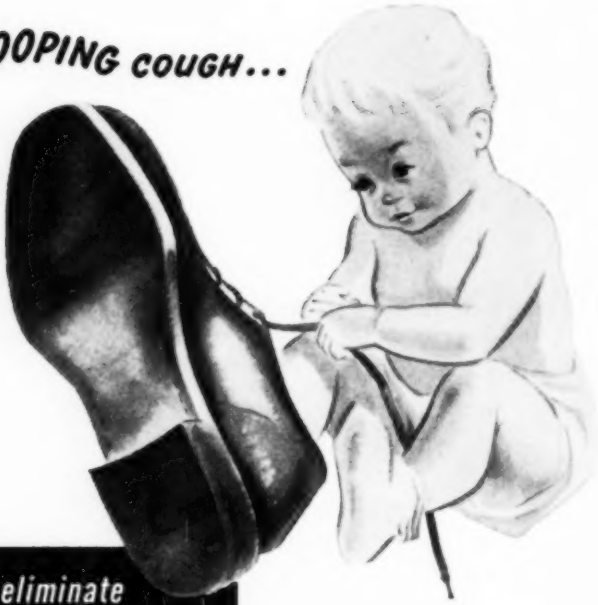

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TETANUS IN AN EPILEPTIC*

REPORT OF A FATAL CASE

H. MELMUTH STERNBERG, M.D.
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AND
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It is generally agreed that tetanus is an intoxication caused by the absorption of a powerful exotoxin produced by the spore-bearing organism, Clostridium tetani; that the toxin readily becomes fixed to nerve tissue and cannot in this state be easily neutralized by specific antitoxin; that following a deep, penetrating wound or extensive burn, a prophylactic injection of antitoxin in adequate dosage will prevent the onset of tetanus. The high incidence of tetanus during childhood, when outdoor activity is great, presents a hazard well recognized by pediatricians and general practitioners. Hence the practice of early immunization against tetanus together with that against diphtheria and pertussis is becoming more and more a standard procedure in preventive medicine. In spite of the use of many new agents in the treatment of this disease, the mortality rate remains high. The difficulties encountered have recently been pointed out by Harris and coworkers.[1] The following presentation is the report of such a case.

CASE REPORT

J. Y., a 10-year-old white male, was admitted to the hospital with a history of stiffness of the neck and shoulders, dysphagia and inability to open his mouth.

*From the Department of Pediatrics, Greenpoint Hospital, Brooklyn, N. Y.

His family, postnatal and developmental history were irrelevant. He had received no immunizations against smallpox, diphtheria, pertussis or tetanus. Aside from whooping cough at the age of one year and tonsillectomy at the age of three, his early childhood was uneventful. His ears had been discharging since early childhood and since four years of age epileptic seizures, once or twice daily, were noted. During these attacks there was urinary incontinence, loss of consciousness and frothing at the mouth. The attacks were preceded by drowsiness and "shakiness." There was no family history of epilepsy or history of trauma.

One week prior to admission to the hospital the patient had an epileptic seizure in the street—fell and struck his head sustaining a laceration of the scalp in the right temporal region. He was brought to the emergency room of a hospital where five sutures were applied. No tetanus antitoxin was injected.

On admission to the hospital he was critically ill, in a stuporous condition, with noisy respiration, shallow and abdominal in character. There was risus sardonicus, trismus and opisthotonus. Pupils were equal and reacted to light and accomodation. The right ear had a purulent discharge and the left showed a chronic otitis media. The heart was normal, its action rapid; the chest was clear. There were no pathological reflexes; the temperature was not elevated. The urine showed heavy traces of albumin and many fine and coarse granular casts. Wassermann and Mazzini tests were negative. The remainder of the laboratory findings included: hemoglobin 11.0 gm.; red blood cells 5.3 million per cubic millimeter; white blood cells 10,100 per cubic millimeter with polynuclear cells 59 per cent and lymphocytes 30 per cent.

The wound in the right temporal region consisted of a laceration of about 1.5 inches with marked swelling of the surrounding area and exuded a serosanguinous discharge. There was also marked local edema of the face.

The wound was excised and debridement performed; deep pockets were opened; the wound was cleaned with peroxide of hydrogen and 10,000 units of tetanus antitoxin injected around the wound; in addition, 100,000 units of aqueous penicillin were injected in the immediate area of the wound. After preliminary skin tests for horse serum sensitivity, 40,000 units of tetanus antitoxin were injected intravenously and the same dosage given by

intramuscular injection. To maintain relaxation of the muscles and overcome the spasms, sodium amytal 3¾ grains was given intramuscularly and sodium pentothal injected intravenously. Paraldehyde in doses of 3 drams and chloral hydrate 15 grains were also employed. The sedatives were not given at specific intervals but only when needed to overcome spasms and rigidity. To accomplish this the patient had to be constantly observed and a close watch was maintained at all times. The injection of curare in the form of intracostrin intravenously had only a fair measure of success and further injections were not attempted.

Alimentation was by nasal tube and supported by 5 per cent amigen and glucose, as well as vitamin C. To prevent secondary infection and inhibit the growth of Cl. tetani, large doses of antibiotics were given in the form of penicillin, aureomycin and streptomycin.

There were daily consultations with the neurological, anesthesia, surgical, as well as ear, nose and throat services. The patient was placed in a quiet, isolated room where light was subdued.

Because of attacks of tonic and clonic convulsions, spasms of the larynx and fixation of the muscles of respiration and failure to relieve pulmonary obstruction by aspiration, Trendelenburg position and oxygen, a trachetotomy was performed and oxygen given under positive pressure. There was continuous 24-hour nursing service with frequent suction and cleansing of tracheotomy tube, changing of position and injections of sedatives to control spasms and convulsions.

On the third day of admission, the temperature rose to 108° F. The breathing was regular and quiet. There were no convulsions. He responded to hydrotherapy in the form of ice water enemas and iced sheets. The temperature dropped to 102° F. in one-half hour. The condition continued unchanged with a variable temperature and an occasional spasm. There were periods of cyanosis and respiratory difficulty requiring aspiration, oxygen and stimulation. On the fourth day of admission, the chest showed diminished breath sounds anteriorly, suggestive of atelectasis due to a mucous plug.

Five days after admission the general condition was good, spasm under control, hydration and nutrition satisfactory, air-way clear; the patient's color was good. Three hours later the child stopped

breathing. There was marked cyanosis and the heart sounds were inaudible. When a thick crust was removed from the tracheal opening, followed by saline irrigation and aspiration of blood tinged mucus, respiration followed and heart action became good. The patient continued on, more relaxed; color and pulse remained good until, ten days after admission, when respirations suddenly became labored and cyanosis very marked. All attempts at resuscitation failed.

SIGNIFICANT AUTOPSY FINDINGS

The body is that of a well-developed, well-nourished white male child. The head shows a recent healing wound of the right parietal region measuring 5 cm.

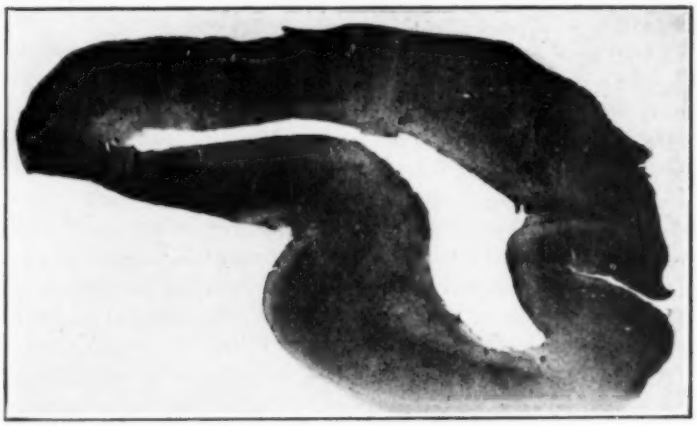

Fig. 1. Wall of cyst showing fibrosis.

Trachea and Bronchi contain thick, tenacious mucoid material.

Lungs. The lungs weigh 400 gms. The left lung is completely collapsed in situ—red, fleshy and sinks in water. The anterior portions of the right lung are reddish and subcrepitant; the posterior portions are red and fleshy. On cut section the parenchyma is red-grey to purplish red—moist, exuding small amount of red fluid. On microsection the alveoli are atelectatic; the intervening septa are infiltrated with cells and fibrin and the bronchioles are collapsed. The bronchial walls show inflammatory reaction.

The *liver* shows cloudy swelling and the *kidneys*, acute glomerulonephritis.

The *brain* weighs 1150 gms., is of normal size and shape and consistency, except for the anterolateral surfaces of the right temporal lobe, which exhibits a firm greyish-white opague ill defined elevated area. On cut section the latter shows a thick walled, smooth lined, partially collapsed cyst filled with gelatinous white material measuring up to 5 cm. in its greatest diameter. The remainder of the brain shows no significant changes.

Sections of the cyst show gliosis of the wall with areas of rarefaction and considerable calcification indicating an old lesion. Occassional very small recent perivascular hemorrhages are noted. Here and there minute spicules of bones are noted in the cyst wall,

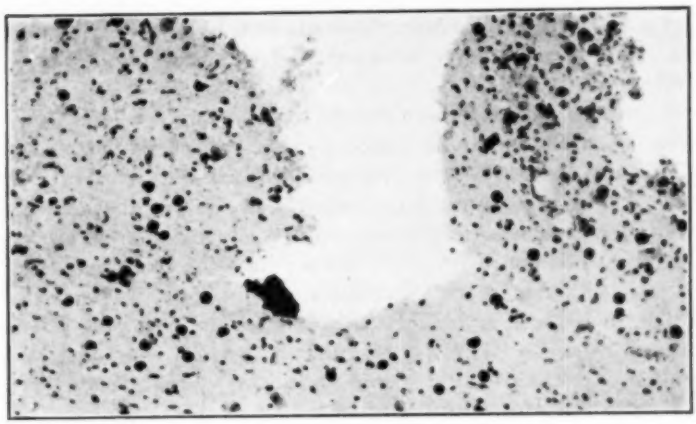

Fig. 2. Wall of cyst showing gliosis and calcification.

between the regional convolutions. Sections of other parts of the brain show congestion and scattered small perivascular hemorrhages both in the parenchyma and leptomeninges.

Cultures of the scalp wound failed to reveal Cl. tetani. Cultures from the cyst and pus from right ear revealed Gram-positive cocci and Gram-positive bacilli.

COMMENT

The failure to receive tetanus antitoxin in this patient after a

wound contaminated by soil and requiring 5 sutures is an indictment of any well regulated hospital. At least 3,000 and possibly 5,000 units should be injected within a few hours after the injury. In severe dirty wounds or when administration has been delayed several days, even 20,000 units are indicated.[2] In individuals who have been adequately immunized actively by means of tetanus toxoid and have received sufficient booster doses, no antitoxin need be given except perhaps in wounds exhibiting severe trauma and massive contamination because the booster injection in these cases may not exert its effect fast enough. According to Miller,[3] the maximum prophylactic effect against tetanus in severely wounded immunized persons may be obtained by simultaneous injection of toxoid and antitoxin in different extremities. The passively induced immunity in these cases does not seem to interfere with the boosting action of toxoid. We[4] have recently stressed the importance of never omitting an "emergency dose" on the slightest signs of injury.

In tetanus immunization the need for a booster dose has always been recognized, since the higher, more prolonged titers produced by the anamnestic response of a secondary stimulus is particularly needed here to counteract the rather sharp fall of the antitoxin titer after the basic immunization. A booster dose of alum-precipitated or aluminum hydroxide-adsorbed toxoid preparation should be injected every second year until the child is 10 years or older. If the child is seen in the interval by a physician for a more serious wound, especially the type of injury associated with tetanus, a booster injection of fluid toxoid should be given at once. This will raise the antitoxin titer promptly—with the exception of the cases cited above—to high protective levels.

The active treatment in our case followed the standard pattern. A single large dose of antiserum is advocated, half of which should be given intravenously. No further injections of serum are necessary and the intrathecal administration of antitoxin is no longer recommended.[5] Perhaps more use should have been made in our case of curare, myanesin and other related drugs. They are now employed in the treatment of tetanus for relaxation of skeletal muscles. Their pharmacologic action is characterized by a blockade of neuromuscular transmission in skeletal muscles at the motor end plates. Careful observation of the patient is essential. The im-

portance of careful nursing to provide adequate nourishment has been pointed out. The alert medical attention of the entire medical and nursing staff of the hospital is just as essential as the cooperation of the medical specialists in the various fields attending to a patient with tetanus.

The most interesting phase of our case is the occurrence of tetanus in a patient with epilepsy. The history obtained indicated that the suppuration in the right ear antedates the onset of convulsions at the age of four. The autopsy revealed a right temporal fully organized cyst of the brain with a calcified wall and a positive culture of mixed bacteria. With the findings of organic pathology in the brain, we have an explanation for the convulsive seizures in this patient.

SUMMARY

A case of tetanus occurring in a child with epilepsy is presented. The cause of epilepsy was a brain cyst as demonstrated on autopsy. Tetanus occurred following a laceration sustained in a street accident. Prevention and treatment of tetanus are briefly discussed.

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OSTEOSCLEROSIS DISSEMINATA FAMILIARIS. (Ugeskrift for Laeger, Copenhagen, 113:929, July 12, 1951). Kristensen concludes that osteosclerosis disseminata familiaris is a benign bone disease with generalized symmetric localization, in some cases accompanied by characteristic cutaneous changes. It is a familial constitutional anomaly transmitted by dominant inheritance. The five cases reported, all in one family, were an accidental finding on roentgen examination for other cause. Cutaneous changes seen in two cases were typical in one case, less characteristic in the other. The blood picture, examined in two cases, was normal, as were the serum calcium and serum phosphatase examined in one case. One patient had vague joint symptoms and muscular weakness of transient kind, another had had rheumatic pain in the muscles since childhood.—*Journal A.M.A.*

HAND-SCHÜLLER-CHRISTIAN SYNDROME*

Report of Case

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The first pathological report of this condition was that published by Hand in the "Transaction of the Pathological Society of Philadelphia," Vol. 16 in 1891-92 under the heading of "General Tuberculosis" and also in the Archives of Pediatrics, Vol. 10, 1893, with the heading of "Polyuria and Tuberculosis." The case to which he refers was a boy of three years with a history of great thirst and polyuria of 8 weeks duration. The child had exophthalmos of both eyes. He died about two months later, and post-mortem revealed the presence of a yellow spot about the size of a five-cent piece near the right orbital eminence. The entire thickness of the bone was soft and movable. Similar small areas, not extending through the vault, were seen on the outer surface. The liver and the spleen were enlarged. Hand at that time believed that these clinical and pathological manifestations were due to tuberculosis, from which the boy suffered.

In 1905 T. W. Kay reported another case under the title of "Acquired Hydrocephalus with Atrophic Bone Changes, Exophthalmos and Polyuria." The case was that of a boy, aged 7 years, whose gums, 5-6 months after scarlatina, began to separate from the teeth. The mother about this time noticed a soft space on the head where there seemed to be no bone. Exophthalmos and polyuria developed. There were similar bone lesions in the lower jaw.

Afterwards, in 1915, Schüller reported two cases, and Christian reported another case in 1920. A further case was described by Hand in 1921. By that time, it was realized that cases represent a bony lesion characterized clinically by extensive defects of the skull bones, exophthalmos and diabetes insipidus. It was also appreciated that diabetes insipidus was related to disturbance of pituitary function, but the nature of the bony lesion still remained in the dark. Rowland, in 1928-1929, through an exhaustive study of the subject, approached the true nature of the mystery. He

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reported 17 cases in young children, and gave a classical pathological description of the condition. He described yellow to brown growths arising from the dura, periostium, pleura and peritonium, which at times assume a tumour-like proportion. Histologically, the lesions were composed of cells of endothelial origin with darkly staining nuclei. Frequently true foreign body giant cells were present and also foamy cells containing various lipoids. In the older lesions there were fibrous changes with almost complete absence of foamy cells but with round cells and eosinophilic infiltration. Rowland regarded the underlying fault to be a disturbance of cholestrol metabolism resulting in a high level of blood cholestrol with consequent deposition of lipoids in the reticular cells. Later it was realized that in most cases the blood cholestrol ranged from 200-300 mg. %, and this fact stimulated a change of the conception of Rowland about the true nature of the complaint. It was then suggested that the primary fault lies in the decreased ability of the reticuloendothelial cells to excrete the normal body lipoids with the formation of lipoid granulomatous masses. The condition was thus classified amongst the disorders of the reticuloendothelial system. The basic foundation thus being laid many cases were reported by various authors from 1930 onwards.

The disease always starts insidiously, the first symptoms usually become manifest in early childhood from the first to the seventh year of life, though occasionally not before adult life. It affects the male sex more frequently and usually selects single members of the family. The initial symptoms are misleading and may be mistaken for other conditions until the classical triad develops. Thus swelling and necrosis of gums with sore mouth and resorption of the alveolar bone with extrusion of the teeth may be the first to attract attention. Likewise, an aural discharge, due to involvement of the mastoid, or a cyst-like swelling on the scalp, may be the earliest symptom. The complete picture may soon develop after these initial symptoms, but at times the various characteristic symptoms may not develop for a considerable period. The defects in the skull appear at an early stage of the disease, and later many other parts of the skeleton may show defects, especially the bones of the pelvis, less often the humerus and femora and still less frequently the scapulae, ribs and vertebrae. The skull in typical cases shows numerous destructed areas of irregular size and shape.

The decalcification and malformation of the sella turcica, which is so important because of its effects on the hypophysis, is usually clearly evident in the skiagram, and clinically there is polyuria which may be extraordinary in degree, and polydipsia of corresponding severity; the specific gravity of the urine is very low, but no other abnormality is detected.

Extensive lacunae in the facial bones have often been seen radiologically, sometimes resembling those of the skull. The changes in these cases are most marked on the lower jaw and are responsible for the loosening and falling of teeth. It sometimes happens that there is increased calcification giving rise to thickening of the bone so that the skull may show areas of great thickening in addition to those of destruction. Exophthalmos is nearly always present, often one sided at first; may be very marked and variable in degree. It results from invasion of the retro-orbital space by the xanthomatous tissue. Cutaneous manifestations have been described in less than one-third of the reported cases. Often the skin is dry and pale, rarely classical xanthomata of the skin are present, but frequently papular and vesicular or hemorrhagic rashes occur. The head is often scantily covered with hair and frequently shows brown-yellow crusts. Among the less frequent symptoms are an increase in the size of the head with the appearance of hydrocephalus. Sometimes symptons of cerebral tumor are caused by intracranial xanthomata. The spinal fluid, however, shows no abnormality. The fundus occuli, cornea and conjunctiva repeatedly show changes of varying nature. Subjective sensations, such as headache, pains in the legs and a feeling of general weakness, may occur and somewhat less frequently mental development is retarded.

Sometimes a period of slight fever is present. There are no, or only slight, changes in the basal metabolism. There are no characteristic blood changes, but in fatal cases the patients show shortly before death, a greater degree of secondary anemia with yellow discoloration of the skin. The viscera may also show changes, such as enlargement of the liver and spleen and changes in the lungs.

The occurrence of lung changes leading to fibroses was described by Rowland in 1926. It causes in the skiagram a mottling like that due to miliary tuberculosis, and in fatal cases a diffuse secondary fibrosis has been found and the patient may show a

typical picture of corpulmonal with terminal heart failure. The cholestrol content of the blood is as a rule within normal limits. Usually the disease is progressive, but the course varies from months to many years. Spontaneous remission of all the symptoms is often observed. The prognosis depends on the number and extent of visceral and nervous lesions.

The help that can be offered to such patients is usually limited to pitressin therapy which controls the diabetes insipidus, and x-ray therapy which causes disappearance of xanthomata with temporary remission of the symptoms but new masses keep on appearing. Insulin in doses of 10 units daily has been recommended to improve the nutrition of the patient. Some good results were reported by Herrmann and Nathan, Gaal, Schoenheimer and Thannhauser by keeping patients on a low cholestrol diet. But Warner-Sperry and Schick could not obtain such good results.

CASE REPORT

G. N. S.; male, aged 6 years, hospital No. 367, was admitted February 15, 1951.

Chief Complaints were polyuria, polydipsia, loss of weight, bulging of both eyes; occasional cough; duration 3 years.

Present History. The present complaint started three years ago, by falling of teeth, followed by gradual protrusion of both eyes. Two months later he was noticed to drink tremendous amounts of water and to void an equally great amount of urine. Two months before admission, the bulging of the eyes was noticed to improve. This improvement was coincident with the appearance of teeth at the sites of those previously fallen.

Past History. Nothing of importance.

Family History. Child was born to young healthy parents. He is the second child in the family and has three brothers all of them are apparantly healthy.

Clinical Examination. Child looks peaceful and quiet. He is underdeveloped, his weight is 15 kilos, his height, 89 cm. He is neither anemic nor cyanosed. There is no enlargement of the cervical lymph nodes and no dilated veins in the neck. His pulse is 100, regular and full. Throughout his stay in the hospital he had had a low grade pyrexia ranging from 37.7° to 38° C.

The Head. Circumference of the head is 18 inches; fontanels are closed; there is no separation of the sutures. There is slight

prominence in occipital region; a small soft area was palpated in the right parital bone.

Eyes are bulging, with wide palpebral fissure. The child sleeps with the eyelids half closed. Pupils are equal and react to light and accommodation. Fundus examination on March 2, 1951 revealed a normal appearance.

Skin shows few scattered papules in the groin and axilla. Pigmented spots are seen more profusely on the abdominal wall. A small yellowish xanthomatous mass, measuring 0.5 x 0.20 cm. is present in the left upper eyelid.

Chest. Respiration rate 20.0 per minute. Chest is pigeon-shaped with rachitic beading, movements are equal on both sides. Trachea is central. There is no impairment in the percussion note. Breath sounds are harsh and vesicular. There are scattered rhonchi and few basal crepitations in the left side.

Heart. Apex beat is seen in the fifth left intercostal space in the nipple line. There is no bulging in the precordial region. No other pulsations are seen. No thrill is felt. By percussion the cardiac dullness is not increased; and by auscultation normal heart sounds are heard.

Abdomen. Looks protuberant and there is divarication of the recti muscles.

Liver. Upper border is in the fifth space in mammary line. The lower border is felt two fingers below the right costal margin.

Spleen could not be palpated.

Central Nervous System. Apart from retarded mentality, no abnormality was detected.

Limbs. No edema, no clubbing of fingers or toes. There is slight enlargement of the epiphyses. Legs are slightly curved; musculature normal.

INVESTIGATIONS

(1) *Urine amount:* The daily output varies from 6.5-9.5 litres.
Appearance: Watery.
Reaction: Alkaline.
Specific Gravity: 1001.
Albumen: Very faint traces.
Sugar: Nil.
Microscopic Exam.: Few red blood corpuscles; few pus cells; few calcium oxalate crystals; no casts or ovoe.

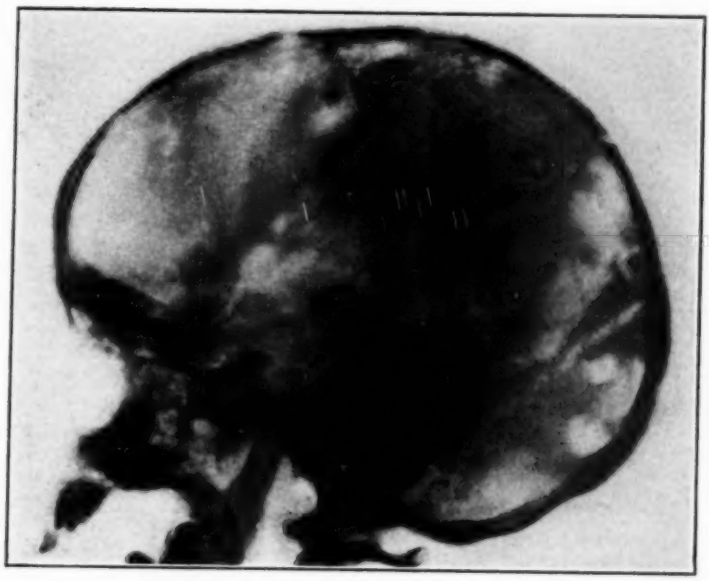
(2) *Stools:* Negative for ameba and cysts.

(3) *Blood Picture* on February 17, 1951: Hemoglobin 120; R.B. C. 5,890,000; W.B. C. 15,300.

Differential Count: Polymorphs 69 per cent; lymphocytes 28 per cent; monocytes 2 per cent; eosinophils 1 per cent.

(4) *Blood Cholestrol* on February 28, 1951: 230 mgm. per cent.

(5) *X-ray Report* on February 21, 1951 (by Dr. M. M. Ragab): "Irregular circumscribed areas of bony rarefaction in the skull, affecting both diploes and taking a geographical

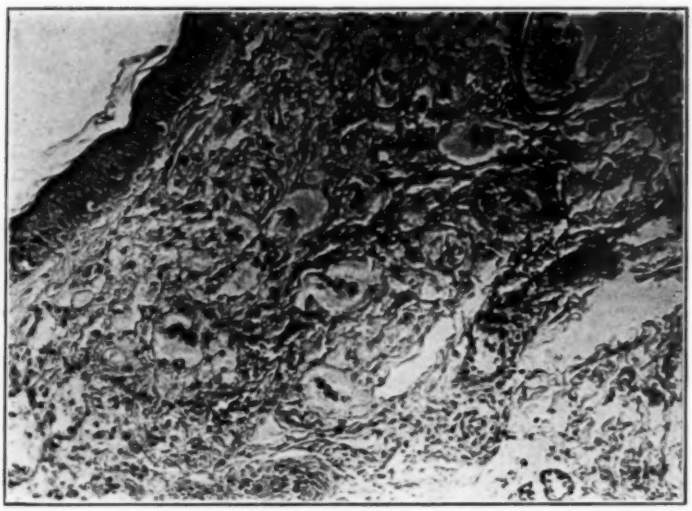


distribution. Some of these areas have punched out edges, especially the occipital bone, and posterior part of the parietal bones. Others show evidence of sclerosis around it especially the frontal bone and the anterior part of the parietal bones. The sella turcica shows small localized areas of bony destruction. The iliac bones also show evidence of irregular rarefaction and coarse trabiculation. Other bones show no changes. Chest shows

evidence of fibrosis in both lung bases. The heart shows no radiological changes.

Conclusion: Radiological appearance is consistent with Christian-Schüller disease."

(6) *Pathological Report* by Dr. S. M. Aidaros: "The material received is a subcutaneous strip of light golden yellow mass 2 x 4 mm. Histologically the covering epithelium is intact all through. The mass itself is made up of numerous foamy cells of various shapes and sizes. Some cells are oval, others are rounded and still a few ones are



polygonal. These cells were previously filled with a lipoid matter the nature of which cannot be ascertained as it was dissolved during the preparation of the paraffin sections. There are also some scattered inflammatory cells of a non-specific nature infilterated amidst the vacuolated cells. Such appearances are compatible with those of a *xanthoma* and exactly resemble those that occur in Hand-Schüller-Christian disease."

Progress of the Case (1) The polyuria improved on pitressin, ½ cc every hour daily. The daily output of urine dropped from 9.5 litres to an average of about 2 litres.

(2) Thyroid tablets, 1½ grains daily for 1½ months, had no effect on the blood cholesterol level.

SUMMARY

(1) This is the first typical case of Hand-Schüller-Christian disease recorded in Egypt. It presents the typical triad of symptoms, i.e., (1) Exophthalmos. (2) Diabetes insipidus. (3) Typical radiological findings in the skull.

(2) It presents also, radiological evidence of fibrosis in both lung bases.

(3) Although the duration of the disease was three years, there were no clinical or radiological evidences of cardiac affection.

(4) Typical pathological pictures of the case are presented.

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DIHYDROSTREPTOMYCIN IN WHOOPING COUGH. (Hospital, Rio de Janeiro, 40: 51, July 1951). Montenegro administered dihydrostreptomycin sulfate to 134 infants and children between the ages of 6 months and 5 years with whooping cough. The drug was given in one daily dose of 0.5 or 1 gm. for seven consecutive days. Sulfadiazine was also given, for the first four days of treatment, in four fractional doses up to a daily dose of 0.5 or 1 gm. In all cases cough diminished in frequency and in violence within the first three days of treatment. Cure, during the week in which the treatment was given, was obtained in 80 patients (60%). In the remaining 54 patients (40%) the disease took a mild course during the first week and was controlled one week after discontinuation of the treatment. There were neither complications nor deaths. The patients gained weight. During 18 months of follow-up, they were in normal health.—*Journal A.M.A.*

THE CARDIAC TRANSITION OF THE NEWBORN

Congenital Cardiac Dilatation With Heart Block

Report of Case

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Part I

Before presenting this case, I would like to deal with the infant mortality, and also with the transition period of the newborn infant.

At the beginning of the century the mortality rate of infants from birth to one year was approximately 99.9 per 1,000 births. If one were to review the mortality infants' statistics of our federal or state departments of health, and of the various mortality records of insurance companies, we would find that this mortality has been lowered to below 50 deaths per 1,000 births, in the first half of the century. This appears to be a very great improvement, and it is. Yet, if one should study the records according to periods or months of life, he would find that the natal mortality and the neonatal mortality up to one month of age has been decreased very little.

Today the records show that between sixteen and seventeen pregnancies per thousand pregnancies end in stillbirth, or spontaneous abortion. The infants that are born alive, between twenty-five to thirty per thousand births, die by the end of the first month. Of this group, approximately six out of every thousand infants die within the first two weeks.

The effects of good prenatal care, and improved technic during the delivery of the infant possibly reflects on the mortality rate, when it is broken down according to areas and population. For instance, in large cities with a population of 100,000 or better, the death rate for infants under one year of age is from 35 to 40 per thousand. In rural areas, the death rate is from 40 to 45 per thousand infants under one year of age. In small cities of 5,000 or under, death often occurs in 50 per thousand infants under one year of age.

In reviewing the causes of these deaths we find that prematurity stands on top, and accounts for one-fourth of all neonatal deaths. Other causes, listed according to their importance, are hemorrhage due to birth injuries, toxic conditions, and to defects and weak-

ness in maldevelopment. Maldevelopments without hemorrhage, especially in the cardiovascular system, the gastro-intestinal system, and in the endocrine system, take their toll. Infection accounts for others, though these deaths have been markedly decreased, due to the present-day antibiotic drugs, such as penicillin, streptomycin, etc., and sulfa drugs.

Being aware of the fact that statistics can be twisted to prove almost any point, I still maintain that these statistics can be twisted any way you want, and you must end up with at least the one and main conclusion, and that is—*too many newborns die*. You must be aware of the fact that natal and neonatal deaths of the first month of age have not decreased to any great extent. To my knowledge they have not decreased more than 5 deaths per 1,000 births in the last 30 years. It is true that, in the one month of age to one year of age groups, great improvement has been made. The big reason for this, however, is a better understanding of the etiology of diseases of this period.

With a better understanding of causes, treatment can be made more specific. In specific therapy, one must give due credit to the antibiotics in the handling of infection. Another big factor in lowering this mortality was the advancement of surgical technic in the field of infant surgery, chiefly in the surgery of the cardiovascular system, and of the gastro-intestinal system. Further advancements have been made in the study of the blood dyscrasias of the newborn and infants in their first year of life, especially erythroblastosis foetalis. Better treatment exists today for infants whose blood picture is complicated by a positive Rh factor. There have also been advances in the diagnosis of the endocrine system, such as persistent thymus, hypothyroid babies, etc., with treatment that gives the infant a good chance of recovery.

It is my opinion that our failure in lowering the natal and neonatal mortality of infants to one month is chiefly due to a lack of knowledge of the true etiology of toxic and physiological conditions, or diseases of the natal or neonatal infant. This is especially true during fetal life. Today we do not have a thorough knowledge of the toxins that are given off by a mother's body to her baby, especially if the mother is affected with a chronic disease, such as diabetes, pernicious anemia, hypothyroidism, or is afflicted with complications in the Rh factor of her blood.

The old belief, that toxins and bacteria do not penetrate from the maternal to the fetal blood, through the desidual membranes and tissues, has been proved false. We know today that the toxins from a disease, such as diabetes, will penetrate the placental barrier, and will have a direct effect upon the development and life of the fetus. This has been amply shown by Herbert C. Miller, who has stated that 30 per cent of all pregnancies complicated by maternal diabetes result in death of the fetus, or the newborn infant. The degree of diabetes suffered by the mother during the pregnancy does not, in any way, influence the results or the effect of the toxins upon the developing fetus. The causes of fetal deaths in infants of diabetic mothers further points to the fact that there is a toxic effect upon the development and physiology of the developing fetus, and the newborn infant.

The first cause of death of infants of diabetic mothers is overweight of the fetus. The second cause is hypoplasia of the islands of Langerhans in the fetal, or infant's pancreas. A third cause is cardiac hypertrophy. This cardiac dilatation or hypertrophy may not be the only cardiac condition found. The infant of the diabetic mother can have this cardiac enlargement complicated by a heart block, from mild to severe.

The main reason for the physician's failure to understand the etiology of the changes and diseases of the newborn is his failure to obtain a thorough knowledge of the functional changes of the physiology of the newborn infant. This is commonly referred to as the transition period of the newborn. It is during this transition period of the newborn that we could save many newborn lives with proper knowledge, by better treatment, and assistance to the newborn while these changes are taking place in the baby's body. The case to be reported in this manuscript, I feel will exemplify this statement.

The effects of the toxemia of a diabetic mother upon the newborn will be shown in the case reported. This mother was a diabetic who was completely under control during her entire pregnancy. She had a normal labor, yet the baby at birth had a dilatation of the heart, and developed a heart block shortly after birth.

THE TRANSITION PERIOD OF THE NEWBORN

The transition period of the newborn infant is that period of

time necessary for the functioning systems of the newborn's body to change from its aquatic life with functional dependency upon the mother, to respirable life in atmospheric pressure and to functional independence. All the physiological systems of the newborn's body go through this transition to a more or less degree. These systems are:

1. Respiratory System.
2. The Cardiovascular System.
3. The Gastro-intestinal System.
4. The Nervous System.
5. The Endocrine System.

Nowhere in the literature have I found any worker who has attempted to classify this transitional period of the newborn. I have given the transition period of the newborn the following classification, as I found that it made it much easier to understand these functional changes. One can also better visualize the functional pathology that results if this transition is delayed or interfered with.

CLASSIFICATION OF THE TRANSITION PERIOD OF THE NEWBORN

First, the Immediate Transition. These are the physiological, functional changes that take place the first hours after birth.

A. The Spontaneous Immediate Transition. These are the physiological or functional changes that take place within the first minutes of life, such as the beginning of respiration.

B. The Prolonged Immediate Transition. These are the physiological or functional changes that take place within hours after birth. An example of this is the contracting down of the muscles of the heart from its dilated size present before and at the time of birth. It usually takes place about one hour after birth.

Second, the Progressive Transition of the Newborn. These are the physiological or functional changes that begin slowly, and progress slowly to their completion, which may be a matter of days or months.

A. The Accelerated Progressive Transition of the newborn are those physiological or functional changes that start at birth, and are usually completed within seven to fourteen days after birth. The permeability of the newborn's gastro-intestinal tract is an example of this type. The permeability is present at its fullest at

birth, and gradually lessens until about the fourteenth day when it disappears entirely, and assimilation of the intestinal contents takes place only by absorption.

B. The Gradual Progressive Transition of the newborn are functions that are present at birth, but change slowly over a time of weeks and months to their completion, when their functions come to normal. This can be demonstrated in the rate of secretions of the digestive engines. These gradually build themselves up in volume, but do not become normal until about the third month.

After the completion of the transition period the infant passes into a Development Stage, and all the organs and structures of the body increase or decrease in size to their anatomical normal. This period usually is between the second and third year.

After this period the infant follows, more or less, a fixed pattern of normal growth.

The report to be given in this manuscript is the failure of transition of a newborn's heart. It is for this reason discussion will be limited to the heart of the newborn.

In order to understand the cardiac transition or the physiological and anatomical changes in the newborn's heart, one must have as thorough a knowledge as possible, of two classes of hearts—first, the heart of the fetus at term; second, the heart of the adult. This is necessary, as one must know what the fetal heart is at the time of birth, and what it is to develop into (that is the adult heart) after the transition period and the period of growth has taken place. If one has this knowledge he can better understand the functional and anatomical changes that take place during the transition period.

THE ADULT HEART

A detailed description of the adult heart will not be attempted. A brief review of the adult heart's gross structures will be given.

The adult heart is a hollow, muscular organ. It is divided longitudinally in the center by a partition called the septum into the right and left heart. The cavities of the right and left heart are again divided by a constriction into upper and lower cavities. The upper cavities are called the right auricle and the left auricle. The lower cavities are called the right ventricle and the left ventricle. The size of the heart is approximately 12 cm. long, and 8 to 9 cm. in

breadth, and 6 cm. in thickness. The heart weighs in the male, between 280 to 340 grams; in the female, 230 to 280 grams. In shape, the heart is an irregular, flattened cone with its long axes from base to apex, running obliquely from behind, forwards, and to the left.

The right auricle is larger than the left, though the walls are thinner than in the left auricle. The walls measure about 2 mm. The volume is about 57 cc. The right auricle can be divided into two parts—the sinusvenosus or the posterior portion, and the auricula or the anterior portion. The openings of the right auricle are the superior vena cava which returns the blood from the upper portion of the body to the heart. There is no valve at this opening. The inferior vena cava is an opening through which the blood returns from the lower portions of the body to the heart. This opening is covered by a semilunar valve which keeps the blood from returning into the inferior vena cava. The coronary sinus is the opening through which the blood returns from the coronary veins to the heart. This is covered by a semilunar valve known as the coronary valve. There is an opening between the right auricle and the right ventricle. This is covered by what is called the auricularventricular valve or the tricuspid valve. There are also many small openings in the surface of the right auricle which are the openings through which blood passes directly from small veins in the heart muscle to the cavity of the auricle.

The right ventricle is a triangular cavity holding approximately 85 cc. of blood. The muscle walls of the right ventricle are much thinner than those of the left, the ratio of difference being approximately one to three. The openings of the right ventricle are the right atrioventricular opening, which, as mentioned before, is closed by the tricuspid valve. The pulmonary opening is covered by semilunar valves of which there are three in number—two in front, and one behind.

The left auricle is smaller than the right auricle. The walls of the left auricle are thicker than the right, measuring about 2 mm. The left atrium is divided into two parts. These are the principal cavity, and the auricula. One opening of the left auricle is the opening for the four pulmonary veins. This opening is not provided with valves. The other opening is a left atrioventricular opening or the opening between the left auricle and the left ven-

tricle. This opening is covered by the bicuspid or mitral valve.

The left ventricle is longer and more conical in shape than the right. The capacity of the left ventricle is approximately the same as the right ventricle. The muscle walls of the left ventricle are about three times as thick as those of the right ventricle. The openings into the left ventricle are the left atrioventricular opening, which is covered by the mitral or bicuspid valve, and the aortic opening, or the opening through which the blood passes from the ventricle into the aorta, then through the systemic circulation. The aortic opening is covered by the aortic semilunar valves, and these valves prevent the blood from being regurgitated from the aorta into the left ventricle.

The muscular structure of the heart consists of fibrous bands which are intricately interlaced. The musculature of the heart comprises first, the fibers of the atria, second, fibers of the ventricles, and third, the auricularventricular bundle of His, or the conduction fibers of the heart.

THE CONDUCTION TISSUE OF THE HEART

The first portion of the atrioventricular bundle of His begins at the atrioventricular node, or the node of Stanley Kent. The node is found at the base of the interauricular septum, at the posterior margin of the right side. It continues forward and downward as a bundle, to the interventricular septum where, a little in front of the septal valve, it divides into a right and left branch. The right and left branches then run down each side of the septum, immediately underneath the endocardium. Each one of these main branches divides into an intricate system of smaller branches which are reflected over the intersurface of the ventricles, and become the Purkinje's fibers of the heart. These branches or fibers end in a very close association with the capillary muscle of the heart. The atrioventricular node and bundle measure about 15 mm. in length, and about 2 mm. in width. Within the muscle fibers of the bundle of His there are minute nerve fibrils which are intimately intermingled with the muscle fibers. The separate nerve filaments are furnished with small ganglia, and these are especially found in the nodes of the heart. The nerves to the heart are derived from the cardiac plexus, and these are formed by branches of the vagi, and from the sympathetic trunks.

THE CORONARY VESSELS

The blood is sent into the muscles of the heart from the right and left coronary arteries which arise in the aorta. The coronary veins carry the blood from the heart muscle and empty into the right atrium.

FUNCTIONS OF THE HEART

The main function of the heart is to circulate blood throughout the entire body. This is accomplished by the pumping action of the heart (with both positive and negative pressures) through three systems of arteries and veins known as:

1. The systemic circulation which consists of all the arteries and veins in the body, with the exception of the lungs and heart muscles.
2. The pulmonary circulation. This consists of the arteries and veins which go through both the right and left lungs.
3. The coronary circulation. This consists of the arteries and veins which extend throughout the heart muscle.

CIRCULATION OF THE HEART

Pulmonary Circulation. The impure or venous blood is received into the right auricle by the superior vena cava and inferior vena cava. On contraction of the auricle, this blood passes through the right atrioventricular valve, into the right ventricle. On contraction of the right ventricle the venous blood enters into the pulmonary artery, and circulates through the lungs. After passing through the capillaries of the lungs, the aerated or pure blood is carried to the left auricle.

Systemic Circulation. On contraction of the left auricle the arterial or pure blood passes through the left atrioventricular valve, and enters the left ventricle. Upon contraction of the left ventricle the blood passes through the aortic valve into the aorta, where it is pumped by the heart throughout the body, with the exceptions of the pulmonary circulation.

Coronary Circulation. The blood enters the coronary arteries in the right and left aortic sinuses. It is returned to the heart by the coronary veins, and enters into the right atrium through the coronary sinuses.

Arteries. The arteries carry the pure or arterialized blood throughout the various circulations, with the exception of the pulmonary arteries which carry venous blood. When leaving the heart the arteries are large, and continuously become smaller until, at their termination, they become arteriols, which enter the capillary system. This system delivers the blood to the veins. The veins carry the impure blood from the capillary system, and return it to the heart. In the pulmonary system the veins carry the arteriolized blood to the heart. After entering the capillaries, the blood enters the venules, and then into the veins that form numerous small branches which carry the blood to larger veins. The large veins enter into either the superior or inferior vena cava which enters into the right auricle of the heart.

ACTIONS OF THE HEART

It is accepted that the chief action of the heart is a pump action. If it is a pump action, then it must be governed by the laws of the pump. The action of the heart is that of a force pump. The force pump has, during its cycle, a positive pressure, a negative pressure, and a period of pause between the strokes of the pump.

This can be duplicated in the human heart, for the contraction of the heart has three stages—first, systolic, which is a positive pressure; second, a diastolic, which is a negative pressure (by negative pressure, I do not mean vacuum); third, the diastasis. This is a period of pause.

Each of the four cavities of the heart duplicate these three stages. Let us apply these principles to each of the four chambers of the heart. We will start with the right auricle, into which the blood returns to the heart, through the superior and inferior vena cava. The blood is carried to the inferior vena cava by the veins of the lower part of the body. Since the blood must rise against gravity, the following physical factors cause the blood to flow toward the heart in the veins.

1. Mechanical:
   a. Valves keep blood from experiencing a back flow.
   b. Muscle action—pressure of muscular contraction on veins forces flow of blood in veins.
   c. Heart action of right auricle—suction action.
   d. Rate of heart beat—tends to even and rhythmic flow.

2. Hydrodynamic:

a. Increased velocity of blood as it enters the large veins.

b. Volume of blood in the veins.

c. Venous pressure, negative in the superior and inferior vena cava as they approach the heart; positive pressure of over 10 mm. mercury in small veins.

d. The greater negative pressure is in the auricles upon the beginning of diastole.

THE RIGHT HEART

After the pause following systole, the auricle begins to dilate. The pressure of the inferior vena cava is greater than the auricular pressure, opening the valve of the inferior vena cava, causing the blood to enter the auricle. The superior vena cava has no valve, so the blood in the vessel is poured into the auricle both by the force of gravity, and the pressure of the adjoining veins. The venous pressure of the adjoining veins has assisted in pushing the blood toward the heart. The suction formed by the negative pressure of the auricle drawing blood from the inferior vena cava, and the rush of blood from the superior vena cava, fills the right auricle with blood.

As the pressure of the blood in the right auricle becomes greater than the pressure of the right ventricle, the right atrioventricular valve or the tricuspid valve opens with the contraction (systole) of the auricle, forcing the blood into the right ventricle, which is beginning ventricular diastole. At the beginning of ventricular diastole, there is a strong negative pressure in the interior of the ventricle, which disappears quickly after the opening of the tricuspid valve (Goltz and Gaule).

When the pressure in the right ventricle is greater than in the right auricle and the pulmonary artery, the tricuspid valve closes, and the pulmonary valve opens, allowing the right ventricle, during systole, to force the blood into the pulmonary artery. As the pressure in the pulmonary artery becomes greater than the right ventricle, the pulmonary valve closes. At the conclusion of systole there is a slight pause in the heart action called the diastaese phase of the heart cycle.

One must bear in mind that the right heart has fulfilled the requirements of a force pump. On the stroke of contractions

(systole), there is a strong positive pressure. On the start of dilatation (diastole), there is a short period of negative pressure, followed by an increasing positive pressure. When systole ends, there is a pause (diastaese). This action can be summed up as follows: The pressure of the right heart consists of a greater positive pressure and a lesser negative pressure.

THE LEFT HEART

The pure blood has returned to the heart from the lungs and enters the left auricle by the pulmonary openings. There are no valves covering this opening. Upon the start of dilatation (diastole) the blood is forced into the auricle by the negative pressure present in the auricle at the beginning of systole, and the positive pressure of the pulmonary veins.

When the auricle is filled and the pressure is greater than in the ventricle, the left auricularventricular valve or the mitral valve opens, and the blood gushes into the left ventricle by the force of the negative pressure in the ventricle at the start of ventricle diastole, and by the left auricular contraction (systole).

Upon the maximum filling of the left ventricle, the pressure becomes greater than in the aorta, opening the aortic valve. Left ventricular contraction (systole) forces the blood into the aorta, and circulates it through the body. After the ventricle empties, the aortic pressure is greater than the ventricular pressure, closing the aortic valve. This completes the heart cycle. Here in the left heart, even more so than in the right heart, the force pump works with a greater positive pressure, and a lesser negative pressure.

CONDUCTION OF CARDIAC IMPULSES

The origin and propagation of the heart beat is much discussed, and unsettled. There are those who hold to the myogenic theory, and others, the nuogenic theory. To me, the myogenic is more possible for the following reasons:

First. The nerves to the heart can be severed, and the heart will keep on with its beat. Second, without the pressure of warm blood flowing over the sinoauricular node, the heart will not contract.

No one, however, disputes the fact that the nerves to the heart have a marked influence on the heart beat. This can be easily

demonstrated with drugs. The rhythmic beat of the heart can also be interfered with by pressure, thermal applications, or drugs upon the sinoauricular node, or the auricularventricular node. Pressure, toxins, and ligations which sever, either partial or complete, the auricularventricular node, will interfere with the ventricular beat, and can produce a heart block, either partial or complete.

MYOGENIC PROPAGATION OF THE HEART BEAT

The blood flowing over the sinoauricular node starts an excitation wave in the muscles of the right auricle, causing the right auricular muscle to contract. This excitation wave is carried to the left auricle, causing the muscle of the left auricle to contract. The excitation wave in heart muscle of the auricle travels about 1,000 mm. per second. Since excitation waves start in the right auricle it is a fraction of a second after the right auricle begins to contract, that the left auricle starts its contraction. This excitation is carried through the auricularventricular node, where it has a short pause, and it is then conveyed to the ventricular muscles by the auricularventricular bundle, to the muscle structure of both ventricles, causing the two ventricles to contract simultaneously.

THE FETAL HEART AT TERM

The fetal heart differs both in anatomy and function from the adult heart. Anatomical differences are pointed out in the following:

Position—higher in thoracic cage, base displaced dorsal by a large thymus gland, aortic arch flat, and more dorsal.

Auricles—in thickness, the right is heavier than the left. In size, anatomy text claims that at birth the ventricles are greater than the auricles. This differs from present x-ray findings. These state that the auricles and appendages are large in comparison to the ventricles. It is my contention that the x-ray findings are more accurate for this reason. The anatomist deals with dead tissue. Not all infants die within several hours of birth, and the pathologist and anatomist viewed the heart after the immediate transition had taken place. Consequently, they would describe the size of the auricles after the contraction of immediate transition has taken place. X-ray evidence points to the fact that the auricles of the newborn do not start to contract to their normal natal size until one hour after birth. If this is true, it is safe to say that

this dilatation of the auricles of the newborn heart are present before birth. In other words, dilated auricles are normal for fetal hearts at term.

FORAMEN OVALE

An opening in the lower part of the auricular septum allows the blood to pass from the right to the left auricles. This is normally present only during fetal life. The valve of the inferior vena cava is much larger in the fetus, and the opening larger than later in life. This opening must be larger, in order to handle the larger volume of blood in the fetus. The enlargement of the valves is formed so as to direct the blood from the inferior vena cava to the foramen ovale. Auricular systole is shorter and auricular diastole is longer than in the ventricles.

Ventricles—in size they are equal. In thickness, the wall on the left ventricle is thicker than the right.

Right and left heart—in size, the right heart is larger than the left heart.

Blood vessels—ductus arteriosis is a short tube or artery forming a continuation of the pulmonary artery to the aorta. Blood vessels at term have a greater diameter than during life, due to the increased volume during fetal life.

Elastic fibers of blood vessels are underdeveloped at term. The lumen is narrower, and thick-walled. Capillaries are larger than awhile after birth.

Placental vessels—the umbilical vein carries blood from the placenta to the liver, and the inferior vena cava. It enters the abdomen and it extends upwards, passing along the free margin of the falciform ligament of the liver, giving off branches to the liver, with the extension of the main vein going to the inferior vena cava, as the ductus venosus.

The right and left umbilical arteries branch from the right and left hypogastric arteries, extend to the right and left of the bladder, then travel to the abdomen where they meet the umbilical vein, leave the abdomen, and extend to the placenta.

CIRCULATION OF PLACENTA AND FETAL HEART AT TERM

The pure or oxygenated blood is returned to the fetal circulation by the umbilical vein. When the vein reaches the liver it sends three or four branches into the liver where the blood is sent to

the inferior vena cava by the hepatic circulation. The remainder and greatest amount of blood passes through the ductus venosus to the inferior vena cava where it is sent to the heart. It enters the heart upon the opening of the inferior vena cava valve, and mixes with the blood from the superior vena cava. From the right auricle it passes through the foramen ovale to the left auricle, and through the tricuspid valve to the right ventricle. The blood is forced from the right ventricle, through the pulmonary valve, and into the pulmonary artery where the greatest amount of blood enters the ductus arteriosus to the aortic arch. A small amount of blood enters the pulmonary circulation to provide blood for nutrition and growth of the lungs.

The blood that enters the left auricle (the major amount comes through the foramen ovale, from the right auricle; a minor amount, from the pulmonary veins) passes through the mitral valve to the left ventricle. Here it is pumped through the aortic valve into the aorta where it joins the blood from the right ventricle for circulation through the systemic system, and also to be returned to the placenta by the right and left umbilical arteries which branch off of the right and left hypogastric arteries.

FUNCTION OF FETAL HEART AT TERM

In the study of the function of the fetal heart at term, we must bear in mind that the fetal heart pumps twice the volume of blood that is required of the newborn heart. It not only has to pump the blood for the fetal circulation, but it must also pump the blood for the placental circulation. The blood vessels of the fetus also accommodate themselves for this extra load by a greater diameter than that present after the transition period.

The placenta has three functions. They are first, fetal nutrition; second, fetal respirations, and third, fetal elimination.

To my knowledge, no worker has attributed any function to the placenta in forcing the blood to the fetal heart. Velocity and flow of blood from the capillaries to the umbilical vein might account for the blood leaving the placenta, but it could never account for its moving along the umbilical vein, which can measure up to four or more feet in length. The blood in its movements in the vein is not assisted by muscle force, or the function of valves.

When the blood from the umbilical vein passes through the ductus venosa and enters the inferior vena cava, it is carried along

to the heart both by the positive pressure in the lower inferior vena cava and the velocity of the blood as it flows toward the negative pressure of the cardiac end of the vein.

Contrary to present day beliefs, it is my impression that the negative pressure of the right auricle, helped by the negative pressure of the left auricle, in their sucking actions, plays the biggest part in the return of the blood from the placenta to the heart. This statement is made because of the following:

1. If the action of the heart is that of a force pump, pumping the blood by positive pressure through the body into the placenta (a closed system), then there must also be a lifting effort or suction (negative pressure) to return the blood to the heart.

2. As previously stated, the auricles at term are comparatively larger than the ventricles, for the heart of all newborn infants are shown, by x-ray, to be dilated.

3. The right atrium is larger than the left.

4. The right heart is larger than the left heart.

5. Auricular diastole is longer, and systole is shorter than in the ventricles. The inferior vena cava has a negative pressure at the cardiac end, and the pressure in the right auricle must be lower than the blood in the vena cava to allow the valves of the inferior vena cava to open. The right auricle has a greater dilatation (much larger than after the immediate transition), causing the negative pressure to exist much longer than it does in the right auricle of neonatal life, that has constricted to its neonatal size.

It is this suction action that plays the greater part in drawing the blood from the inferior vena cava and umbilical veins. The blood from the superior vena cava, due to the cephalic presentation of the majority of fetuses, cannot be acted upon by gravity to pour the blood into the auricle. The velocity and flow of blood of the superior vena cava is also helped by this suction action of the right auricle.

Blood also flows into the left auricle through the foramen ovale, both during systole and diastole. For this reason, the right auricle dilates more than the left, as the right auricle must fill two chambers of the heart—the left auricle, and the right ventricle. This requires a greater volume of blood.

It is my opinion that, in this greater dilatation and lesser contraction of the right auricle, the pump action of this chamber of

the heart functions with *a greater negative pressure and a lesser positive pressure.*

Though the left auricle does not dilate to the extent of the right auricle, its dilatation is much greater than is the contraction. The greater negative pressure is caused by this greater dilatation, and helps draw the blood into the left auricle. The left auricle is also functioning on a greater negative pressure.

To further justify this statement (which I know will be very much questioned), let me present:

It might be stated that this dilatation is due to the greater volume of blood in the fetal circulation (Twice that of the heart of the newborn). If this is the case, then one would expect to find a similar dilatation in the ventricles, but you do not. It is the laws of the force pump that controls the flow of blood through its chambers.

In the embryonic development of the heart from the time the heart becomes a four chanbered pump, the auricles develop into much larger cavities than do the ventricles, and remain as such until one hour or more after birth. If the fetal heart was circulating the blood through the fetal and placental circulation on a greater positive pressure, then you would expect to find the auricles smaller than the ventricles, as in life after the completion of the transition period.

Further, the ventricles are both the same size in the fetus at term. Both the right and left ventricles have a similar outlet into the aorta. The right, by the ductus arteriosus; the left, through the aortic opening. Their size depends on the volume of blood they receive, and the force of their pumping action.

Both right and left ventricles are pumping against the same pressure—the pressure of the blood in the aortic artery.

The force and volume of blood entering the right and left ventricles from their respective auricle is equal. The foramen ovale, being open upon contraction of the auricles, their pressure balances, for an enclosed fluid, when acted on by an outside force, exerts its pressure equally in all directions. Similarly, when the atrioventricular valves are closed, and the auricles begin to dilate, the negative pressures of both auricles tend to balance.

The only other way the left auricle receives blood is by the pulmonary veins, but only the blood needs of pulmonary nutrition

and growth circulate in this system. This is not a great amount, and does not influence, to any marked degree, the size of the left auricle.

There is also a negative pressure in the ventricles at the beginning of diastole, which assists the auricles in emptying. The fetal auricles have a lesser systole than the ventricles, so this negative pressure or suction of the ventricles must play a part in their filling.

It is because of these reasons that the following conclusion is given: *the fetal heart at term functions with a greater negative pressure and a lesser positive pressure.*

If this supposition has basis in fact, then the cardiac transition of the newborn is a change from *a greater negative pressure and a lesser positive pressure,* to an *increased positive pressure and a lesser negative pressure in natal life.*

VALVES OF THE FETUS AT TERM

The valves of the fetus at term function the same as the valves of the neonatal infant.

CONDUCTION SYSTEM OF FETUS AT TERM

The conduction system of the fetus at term is fully developed and functioning. There is this difference in its function. The cardiac nerves and the conduction system of the heart of the natal and neonatal infant, being composed of young, highly specialized tissue, are thought to be more sensitive to toxins and stimulation than is the similar system in the adult.

THE CARDIAC PHYSIOLOGY OF THE NEWBORN

This physiology is one of change; a true, physiological change in the newborn system, making it possible for the newborn to live independent of its mother's body, and in a new environment. This is the transition period. While recording these physiological changes, an attempt will be made to classify them by placing their classification in square brackets, according to the classification given earlier in this manuscript.

Upon birth, one of the first physiological changes to affect the heart is the onset of respiration. This is thought to be brought about by evaporation of the skin, with the skin being stimulated by contact with the air, causing the infant to take a deep breath [spontaneous immediate transition].

On the infant's first breath, the chest is expanded, the diaphragm descends, the negative pressure of the thorax becomes greater, combining with the rush of air into the lungs caused by atmospheric pressure, forcing the blood into the pulmonary system [spontaneous immediate transition] by contraction of the right ventricle.

Upon the clamping of the umbilical cord, there is a cessation of flow in the umbilical vein, and the umbilical arteries [spontaneous immediate transition]. If the circulation in the umbilical cord is stopped by a mechanical clamping, we have the spontaneous immediate transition. If the circulation must cease after the delivery of the placenta, without interference, then the flow of blood is stopped by the prolonged immediate transition.

The umbilical veins and ductus venosa are obliterated from three to seven days [accelerated progressive transition]. The umbilical vein becomes the ligamentum teres [gradual progressive transition]. The ductus venosa becomes the ligamentum venosum [gradual progressive transition].

The hypogastric arteries become obliterated from the second to the seventh day [accelerated progressive transition]. These arteries become the lateral umbilical ligaments [gradual progressive transition].

When the placental circulation is shut off from the fetal circulation, fifty per cent of the blood volume of the infant heart is removed. This results in the infant heart performing only one-half the work it had done before birth.

The force pump action of the heart must adjust itself to the change in circulation, and volume. The volume of blood to pass through the left auricle is markedly decreased. The distance the blood of the inferior vena cava is to be lifted in the new circulation has been reduced to only the blood in the inferior vena cava itself or the blood from the systemic circulation. The lift action of the pump, or the suction action of the right auricle, must be greatly reduced, and the positive pressure action of the right auricle markedly increased.

This is accomplished by the lowered pressure needed in the right ventricle to pump the blood through the pulmonary system. The right ventricle no longer must force blood against the stronger pressure of the aortic or systemic circulation. This change in pressure is not spontaneous, but a prolonged immediate transition.

As a large volume of blood is brought to the left auricle by the pulmonary veins, causing a marked decrease in negative pressure (and blood also entering the left auricle by the foramen ovale), the positive pressure of the left auricle becomes greater than the right, forcing the valvular folds of the foramen ovale over the opening, closing it during systole. The closing of the foramen ovale is an accelerated progressive transition sealing off permanently by the tenth day. The final organization of these valvular folds into the auricular septum takes place by the gradual progressive transition, and may be a matter of months.

One more change is necessary before the circulation of the right and left heart become independent systems. That is the closing of the ductus arteriosis, or the ductus Botallo.

Upon expansion of the chest with respiration, the decreased flow of blood through the duct, and the increased pressure in both the pulmonary artery and aorta, and the looping of these arteries (Swindle) to their maximum with each heart beat, the ductus arteriosis is caused to elongate or stretch, closing the duct by bringing the endothelium of the walls of the vessel to come together with pressure. The endothelium of the vessels are made to adhere and fuse together. This is usually accomplished within ten days [accelerated progressive transition]. The duct then undergoes fibrotic changes, and forms the ligamentum arteriosis [gradual progressive transition].

After the first hour of neonatal life, there is an actual reduction in the size of the heart. This reduction is chiefly in the reduced size of the auricles, due to the change from a greater negative to a greater positive pressure [prolonged immediate transition].

The average weight of the heart at birth is 20 gms., and the heart gains very little in size or weight the first six weeks (Scammon).

With the growth of the infant, the position of the heart of the newborn changes. During growth the heart elongates, and descends in the thorax, and the long axis becomes oblique [gradual progressive transition].

CHANGES IN THE AURICLES

At birth the right auricle is heavier than the left, but by one month of age both auricles are equal [accelerated progressive

transition]. After this time there is a marked thickness of the musculature due to the increased contraction of the muscle. In this process of muscle growth, the left auricle becomes thicker than the right, but the right atrium becomes larger in size [gradual progressive transition].

CHANGES IN THE VENTRICLES

Both ventricles at birth are equal, but with the changes of circulation and pressure after birth, the left ventricle becomes larger than the right, and the musculature thickens. At three to six months of age the left ventricle musculature is two to three times thicker than the right (Hecht) [gradual progressive transition].

The muscles of the left ventricle become hard, and those of the right ventricle, spongy [gradual progressive transition]. The remainder of the development of the heart is namely one of growth.

An effort has been made to make this presentation as brief as possible, by only giving what were considered the important factors in the cardiac transition of the newborn. For this reason this treatise should not be considered a complete record of all the detailed changes in the newborns' cardiac physiology. In the presentation of the following case of failure of cardiac transition, one must keep in mind the facts presented in this manuscript in order to understand the reason given for the pathology present.

(*Part II, in conclusion, will follow in April issue*)

PEDIATRICS HALF A CENTURY AGO

From time to time the Archives, which was the first Children's Journal in the English language, will reprint contributions by the pioneers of the specialty over fifty years ago. It is believed that our readers will be interested in reviewing such early pediatric thought.

CHOREA; WHAT IS IT?*

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To answer the questions propounded in this interrogatory title should require a discussion of: (1) Origin and fitness of the term chorea. (2) Classification. (3) Clinical picture. (4) Diagnosis. (5) Pathological Anatomy. (6) Pathogenesis.

Chorea is a term derived from the Greek meaning dance. Paracelsus first applied it to an epidemic disorder of movement which developed in children and adults in Germany and the Netherlands during the fourteenth, fifteenth and sixteenth centuries. For relief of the mysterious disorder people sought the shrines of favorite patron saints. The chapel of St. Vitus in Zabren acquired such a reputation for cures that the Strasburg magistrates ordered the sufferers to be sent to this chapel to be healed by the saint. This affection which was evidently an hysteria or imitation psychosis was also described by other writers and cures ascribed to various shrines, hence the names St. John's and St. Anthony's dance. The disorder was remarked by many authorities and indeed was so extensive as to be referred to as the "procession of jumping saints." The origin of the malady was undoubtedly in an ultra-religious enthusiasm. In our own country in the states of Kentucky and Tennessee at the beginning of the past century, and as the result of religious enthusiasm, it is said that a real Chorea Sancti Viti appeared.

Hecker, in his work which is still the standard authority upon the epidemics of the middle ages, made a special study of the dancing psychoses and discussed them under three heads. With our present knowledge we are able to properly place all of his

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cases under well recognized subdivisions of the various neuroses, or psychoses, and positively differentiate them from chorea.

It is peculiarly unfortunate that the word chorea should have been used by the great Sydenham to designate the rather common disorder which he was the first to carefully observe and adequately describe, and to which, in the following centuries of usage, it has inseperably attached itself. Notwithstading the unfortunate misuse of the word by Sydenham there can now be no valid reason for abandoning it, especially since the epidemic forms of socalled "hysterical chorea," or the "dancing manias" have disappeared. The persistent misuse of this word for centuries in the description of a clinical type, the pathological origin of which is still very obscure, has many parallels in medicine, of which tabes is classical. In the later instance it is to be observed that while our pathological anatomy is exact and extensive, the pathogeny is largely speculative. Tabes, meaning wasting of the back, commits us to no pathologic doctrine nor to the equivocal dependence upon a prominent symptom which in atypical cases may be wanting. Progressive locomotor ataxia is not a good term for not only is the affection oftentimes not progressive but ataxia may not be present; and in the use of the term chorea we commit ourselves to no pathologic doctrine; but, unfortunately, do commit ourselves to a symptom—dancing—which is rarely or never present.

Paralytic chorea, paralytic dancing, limp chorea, limp dancing! It is both ludricrous and amusing and I can imagine no reason for the long continuation of this example of philological sinning than a worthy desire to err in the company of the original nosological sinner, but wonderfully great Sydenham.

It does seem strange, however, that chorea should be made the nosological hook upon which authors and observers have indiscriminately hung all sorts of affections which are characterized by coarse, incoordinate trembling and irregular purposeless movements. Apparently it has been necessary only for some enthusiastic medical observer to remark a patient with incoordinate muscular movement appearing in any part of the body, to immediately burden medical literature with a new nosological monstrosity like chorea scriptorum, dancing writers' cramp. Think of it! Shade of Paracelsus! Indeed we find in one of the popular medical dictionaries ninety-four separate affections described as sub-

divisions of chorea. This perpetuation of an error causes one to slyly suspect that both ancient and modern physicians were better clinicians than philologists.

To facilitate discussion and study of chorea, Osler has proposed the following four grand divisions:

"Chorea Minor—Sydenham's chorea—an acute disease of childhood, rarely of adults and of the aged, characterized by irregular involuntary movements, a variable amount of psychical disturbance, and associated very often with arthritis and endocarditis. The disease is usually regarded as a neurosis, but the clinical characters of the severer cases, and the frequent heart and joint implications have suggested to many recent writers that it may be due to a specific poison.

"Chorea Major—under which term are now embraced both dancing mania and the various forms of rhythmical or hysterical disorders of motion. Psychical impressions, emotional disturbances, and imitation, play the most import rôles in this form.

"Choreiform Affections or Pseudochoreas—the various forms of habit spasm or tic, local or generalized, which are perhaps best grouped under the latter term, in the more extended use as employed by the French.

"Secondary or Symptomatic Choreas—chronic disorders of motion, which depend upon degenerative and irritative lesions of the motor cortex or path. Here may be included the pre- and the post-hemiplegic disorders of movement, the socalled spastic choreas, and many of the cases of congenital and chronic chorea. One malady alone in this group may be separated as an independent affection, viz., the chronic progressive form, the socalled Huntington's chorea."

The chorea major of this classification should no longer be retained, since it is perfectly possible with our present knowledge to correctly describe each and every one of these conditions as an hysteria or a psychosis. Rejecting the term chorea major as unscientific and misleading it necessarily follows that we should reject the qualifying adjective minor in his first division; and since tic convulsif or Gilles de la Tourette's disease is a well recognized clinical entity, and the localized tics or habit spasms, as we shall later see, differ so essentially and completely from the affection included in Osler's first group that his third group,

choreiform affection or pseudochorea has nothing to recommend it. It is complicating, misleading and unscientific. His fourth group, secondary or symptomatic chorea, is decidedly objectionable but with our present knowledge must remain. Particularly is this true since we adopt the word chorea as an expression of the idea of which it has so long been a sign, irregular, incoordinate and more or less uncontrollable muscular movement; we thus consent to the further perpetuation of an error, respectable and hoary with the sanction of five centuries of doctors.

I believe that you will agree that for convenience of discusion we are warranted in dividing chorea into two classes: (1) Chorea (Syndenham). (2) Secondary or symptomatic chorea. Under the latter I would include: (*a*) Chronic progressive chorea, or Huntington's chorea. (*b*) Pre-hemiplegic disorders of movement. (*c*) Post-hemiplegic disorders of movement. (*d*) Congenital chorea, more properly congenital athetosis. (*e*) Chronic chorea. In the following discussion, chorea (Sydenham) will alone be considered.

Chorea is an infectious neurosis of the developmental period of life, but does rarely occur in the adult and still more rarely in the aged. It is marked by irregular, unintentional and uncontrollable muscular contractions of a complicated nature, and by varying departures from the individual's mental and psychic standard. It is an affection of more or less definite onset, course and limitation, is not infrequently associated with infectious diseases, particularly arthritis or endocarditis, and while it has been commonly looked upon as purely a neurosis, it is now believed that its resemblance to the acute specific infections is so great as to warrant its being considered as such, or the reaction of a susceptible nervous organization to the toxins of various infections. Indeed, Sydenham recognized this saying, "Now this affection arises from some humor falling on the nerves and such irritation causes the spasms." We have not improved upon this pathology advanced by the great clinician two hundred and ten years ago.

FORMS.—In the discussion of the clinical picture of chorea it is best to make a division of the subject, recognizing, first, a common form; second, a grave form; third, a paralytic and fourth, a gestational form.

(1) *Common Form.* The affection usually develops insidi-

ously. The prodromal period is commonly unobserved, but is important. It usually consists of sleeplessness, mental and psychic lassitude, emotional outbreaks, constipation, a capriciousness or lack of appetite, complaints of fatigue and joint pains. Later the child becomes restless, awkward, inattentive, and apparently careless. The teacher often complains to the parents that the child cannot fix its attention, is unable to sit still, that the copy book is blurred and writing slovenly. Oftentimes the child appears not only dull but wilful, fretful, emotional and irascible. Little girls not infrequently complain of their clothing being wet by uncontrolable jets of urine, and frequent urination is common. At home the child seems awkward in gait, drops its knife or fork while eating; lets fall various articles while attempting to handle them. I have heard these children called "butter fingers" by their mothers. Indeed so many awkward catastrophies occur that an impatient parent may, in ignorance of the condition, chastise the child, when suddenly there will burst forth a shower of muscular phenomena, grimacing, shoulder shrugging, finger twisting, and even complete loss of power to walk, stand, or lie, because of the violent, irregular, uncontrollable, and diversified muscular spasms If the disease progresses without such an emotional climax the involuntary and uncontrollable movements insidiously begin in various parts of the body, often affecting the musculature of the face producing all sorts of facial contortions. Smacking noises, especially while eating are produced by the incoordinate and spasmodic musculation of the lips, cheeks and tongue. In addition to the spasmodic twitchings there is an exaggeration of all muscular movement and a diseased condition is recognized.

When brought to the physician a peculiar, awkward, motor restlessness is manifested; even when requested, the child is unable to sit still; the forearm is adducted and abducted or rotated; the fingers are extended and separated and again immediately flexed and extended; the wrists are not often flexed or extended but the shoulders are hunched up and in turn dropped. This shrugging of one or both shoulders is quite characteristic and constant. The child twists and flops about in its chair; the head is twisted from one side to the other; the tongue is protruded with a jerk and retracted in the same way, and followed by the teeth snapping together. Because of the muscular spasm the tongue is often twisted

and rolled about, especially when protruded, and the same causes produce a deepening of the longitudinal furrow, not infrequently so marked as to give the dorsum the shape of the letter v. Repeated sighing, noisy and irregular respirations are common, and in some of the severe cases phonation may be disturbed. When the child is asked to pick up a pin, he or she grabs for it without grace or precision and usually reaches beyond it; the hand is then drawn backward, the pin fumbled, and in a grotesquely awkward manner picked up. In the severe cases the gait is altered; contractions of the muscles of the legs and thighs may become so marked as to prevent walking and even standing. The movements, except in extreme cases, cease during sleep. It is characteristic of chorea that the involuntary movements do not confine themselves to definite muscular groups, nor do they occur with any rhythm, but are purposeless, uncontrolable, and undirected, lacking that delicate accuracy and precision which is the grace of youth and poetry of childhood.

At first the affection usually involves one arm, later extends to the other or to the leg upon the same side, occasionally the face is first attacked, more rarely one leg. It may remain limited to one side only and is spoken of as hemichorea, but in the majority of cases it extends to other members and becomes generalized. Voluntary movements increase the muscular twitchings. These incoordinate spasms combine with the voluntary movements and modify them in such a way as to produce great awkwardness, especially when any finely coordinate act is attempted. Besides the manifest mental and psychic excitability the child learns from experience that, in order to execute any voluntary movement, it must be made as quickly as possible, and in those moments when the choreic twitchings have been volitionally repressed. If the movement is long continued and requires precision and accurate coordination, the choreic spasm surprises the musculature as it were, and converts the normal graceful action into jerky, bizarre movements, oftentimes absolutely defeating the volitional purpose. Continued coordinate effort is difficult or impossible, not alone because of the spasm but from an actual muscular weakness.

Mental change even in the mild cases is nearly always present, and consists in inability to fix the attention, stupidity or irritability and usually a general weakening of the mentality. Without ref-

erence to a true psychosis which may appear in the chorea as a result of a degenerate organization we often observe changes of the psyche even in the common forms. The facies of the sufferers are peculiarly stupid and inane; they become peevish, perverse, deceptive, filthy, and cruel to younger children. I have observed a complete and regretable change in the disposition of a child, due wholly to chorea.

Course: The course of the disease, whether it be insidious or abrupt in onset, is commonly marked by exacerbations and remissions of varying length and degree. Many cases recover without any treatment whatsoever. The average duration of the disease is from six weeks to four months. Occasionally cases are observed to recover with or without treatment in so short a time as two weeks, while others will often persist, notwithstanding the greatest care and skilful medication, for one or two years. Complete recovery is the rule; relapses, however, are extremely common, and death has been known to occur. Rarely the disease becomes chronic and may persist the life-time. Tic and habit spasm not infrequently follow true choreic attacks.

(2) *Grave Form.* Chorea gravis is ordinarily of acute and intensive onset, and as such may be a severe exacerbation of the common type, or appear as a recurrence of either type. It is characterized by intensification of all the motor, mental and psychic manifestations referred to in the description of the common form. The choreic movements are general, widespread, continuous and violent, usually preventing the patient from standing, walking or even sitting in a chair. I have twice observed cases so severe that a bed with padded side rails was necessary to keep them from being thrown to the floor by the violent contractions. In these cases the movements do not stop during sleep as in the mild form, but oftentimes interfere or prevent it entirely. Speech is usually interfered with, indeed they may become completely aphasic. Mastication and swallowing are difficult or impossible. In the uncared-for cases bruises and excoriations, followed by abscesses and erysipelas, may result. A low fever is always present and may become marked. Mental and psychic effects are so grave that the patients may no longer orient themselves. Delirium with hallucinations is common, indeed, maniacal excitement may appear, but a confusional condition is the rule. Acute par-

anoia has been observed. These psychic changes usually develop at the height of chorea and last but a few weeks, and are followed by depression, apathy, or perhaps a degree of mental confusion, or fixed ideas of persecution, self-reproach, etc. These cases so marked by psychic depression constitute the chorea insaniens.

(3) *Paralytic Form.* The paralytic form of chorea, sometimes referred to by English writers as "limp chorea" is not uncommon. In all varieties of chorea there is more or less weakness of the muscles involved, but this weakness is subordinate in comparison with the more noticeable muscular twitchings. It appears that the choreic poison in the paralytic form expends itself in the production of extreme muscular weakness rather than spasmodic contractions. It seems more likely to me that the muscle is so weakened by the action of the poison upon the motor apparatus that it is no longer able to respond either to the morbid impulses characteristic of the disease or to the stimulus of the will. The paralysis may be monoplegic, hemiplegic, or paraplegic. Although the authorities state that monoparesis is most common, my own experience is that hemiparesis is more frequently met with.

The diagnosis of paralytic chorea is not always easy. As a rule it occurs in children of seven or eight years of age, and may be either insidious or sudden in onset. The reflexes on the affected side are nearly always abolished, are never increased on the opposite side, are occasionally diminished, but are rarely completely abolished. Normal myotonia is absent, nor can the reaction of degeneration be demonstrated. There is no pain or tenderness of the paralyzed members nor is there any loss of sensation. Muscular atrophy is very, very rarely present, and when present most likely due to some organic nervous affection as myelitis or neuritis accidentally complicating the disease.

(4) *Gestational Chorea.* The chorea of pregnancy is most common in young primiparæ and those who have been subject to the disease in early life. It may appear at any period of pregnancy, but is most frequent in the first half. This form of chorea varies in intensity but is usually severe and more liable to be followed by mental and psychic changes than other forms. The motor agitation is ordinarily intensive. It may result in premature expulsion of the child, and finds relief in parturition or abortion; on the other hand quite a number of cases fail to be so benefited.

It may take on the paralytic form, and that it is more grave than the ordinary form of chorea is evidenced by statistics which show that 20 per cent of these cases prove fatal.

I have recently observed a patient of Dr. F. W. Linn that presented a paralytic chorea affecting the right upper extremity and in which the ordinary involuntary movements were lacking; the volitional movements, however, were weakly, incoordinately, and awkwardly executed. This case was seen but two days preceding her confinement at term; the chorea immediately ceased after labor.

The diagnosis of chorea is ordinarily thought to be a simple matter. Considerable observation in both general and special practice inclines me to the belief that not over 50 per cent of the mild forms of chorea are treated by the physician. Those affected with the undeveloped forms of the disease attend school and are punished and insulted by the teachers who look upon them as merely restless and irritable children; they are scolded at home, and even when the physician makes the diagnosis they get small sympathy. The majority of them go untreated. General practitioners in and out among the families of their *clientèle* neglect these children and fail to urge upon the parents the fact that the child is suffering from a serious ailment and should receive consideration, rest and medicine.

Physicians heretofore, and at present too often, have very nebulous ideas as to what constitutes a true chorea and the so-called pseudochoreas, hysterical choreas, etc., throughout the whole ninety-four more or less fanciful and certainly erroneous subdivisions of the subject.

It seems to me that the simple classification adopted in this paper and a conception of the disease as outlined in the definition here proposed will very largely do away with the difficulties of a differential diagnosis. There should be no difficulty in differentiating the incoordinate movements of chorea from the rhythmic tremblings of metalic or toxic poisoning, of hysteria, paralysis agitans, and multiple sclerosis. The peculiar movements of Friedrich's ataxia, athetosis, the unrhythmical muscular trembling and incoordinate movements occasionally observed to precede and follow hemiplegia, since each has its own individual signs and clinical picture so well developed that no one should err.

Huntington's chorea is an affection of late adult life, possessing almost universally a history of hereditary transmission and attended by progressive mental deterioration. The myoclonias and socalled electrical choreas and hysterical disturbances of movement resembling chorea present only a superficial resemblance to the disease under discussion. The tics, particularly, the habit spasms, because they not infrequently develop on a choreic basis or remain as relics of choreic attacks, seem to be the ones that cause the great confusion. This is so remarkable that one not infrequently reads long accounts of the cure of these cases under the title of "The Cure of Chorea."

Stevens and Ranney attempted to demonstrate that they cured large numbers of cases of chorea by correcting refractive errors, and of all of the 12 cases which were the basis of one of Ranney's reports, Osler states that not one of them was chorea, but that they were all either habit spasms, tic, or hysterical chorea. And the 5 cases which Stevens presented to the New York Neurological Society as chorea from eye-strain were examined by the members of that celebrated society who were unanimous in the statement that, "not a single case was true chorea." It is merely a question of mistaken diagnosis which ought not occur under any circumstances.

PATHOLOGICAL ANATOMY.—Since Sydenham stated that chorea "arises from some humor falling upon the nerves and such irritation causes the spasms," there have been many theories containing both facts and fancies advanced for its explanation, and today, after many wanderings in a wilderness of speculation, we have practically returned to this pathological opinion which was forcibly expressed by the great Englishman two hundred and ten years ago.

Nine years ago Osler stated, "there are no characteristic lesions in fatal chorea." That is the position occupied today. The literature of the subject contains not a few records of postmortem findings by able pathologists, all of them presenting, however, almost irreconcilable differences. I shall not pursue the subject farther for I feel that your time should not be taken up by a profitless discussion which begins with the unknown and ends nowhere.

Pathogenesis: A satisfactory presentation of the pathogenesis of chorea in the present state of our knowledge is also im-

possible. It is evident, however, that in chorea we have to deal with an abnormally susceptible nervous system and the action of some toxin or toxins which are the result of bacterial infection. The intimate relationship which seems to exist between rheumatism and chorea has been too well demonstrated by clinicians to be further subjected to criticism or denial. Just what this relation is we are at present unable to state. It is very possible that as Eshner states, in the disease which is commonly designated rheumatism there are several clinical entities, dependent upon a number of causative factors. While the actual causation of chorea yet awaits discovery, the belief is growing that the disease is due to irritation of the motor mechanism, most probably the cells of the cerebral cortex, by some irritant of infectious origin. That individual predisposition usually exists is certain, and that the rheumatic diathesis, particularly when associated with degenerative types and precocious development of the mental and psychical functions, must be considered important causative agents is positive. Indeed, it must be regarded as a disease of neurotic stock, a stock too finely organized to safely undertake the competition, the overwork, anxiety, and successes as well as the failures of school life; unable to successfully withstand the changes of adolescence as well as early pregnancies; in fact anything which produces a lowering of the normal physiologic resistance.

Fright as a causative factor, when carefully analyzed and observed, nearly always resolves itself into a climax in the course of the disease already established in a susceptible nervous system, and merely brought to full bloom by the influence of a powerful emotion. Peripheral irritations and the socalled reflex irritations such as worms, eye-strain and adherent prepuce play no greater part in the etiology of true chorea than poor hearing and ingrowing toe-nails. I believe that beyond this very meagre and incomplete discussion of the subject, our present knowledge does not warrant us in going.

DEPARTMENT OF ABSTRACTS

SABIN, A. B.: TRANSMISSION OF POLIOMYELITIS VIRUS; ANALYSIS OF DIFFERING INTERPRETATIONS AND CONCEPTS; PRACTICAL IMPLICATIONS. (Journal of Pediatrics, 39:519, Nov. 1951).

The most significant source of virus is human feces derived from patients and healthy carriers. The period of communicability cannot be said to be limited to the period of acute illness, but should rather be regarded as potential during the period that virus is generally eliminated in the feces. There are many methods by which stool-borne virus can be transmitted and one method cannot be emphasized over another. From the evidence on hand the following measures which are sometimes advocated during epidemics of poliomyelitis are not warranted: (1) Avoidance of crowds. (2) Exclusion of children under 16 years of age from movies, churches and schools. (3) Exclusion of poliomyelitis patients from general hospital wards. However, it appears reasonable during epidemic periods for individuals to take the following simple steps to diminish the risks of acquiring infection: (1) Keep fingers out of mouth and wash hands before eating. (2) Keep flies away from all food, and thoroughly wash that which is consumed uncooked, e.g., vegetables and fruits. (3) Keep children under 16 out of crowded, public wading and swimming pools. (4) Avoid intimate association (shaking hands, kissing, common eating utensils, towels, etc.) with members of a family in which a case of poliomyelitis has occurred within three weeks, even though the patient is in the hospital.

MICHAEL A. BRESCIA, M.D.

NEWTON, W. A. AND ZUELZER, W. W.: IDIOPATHIC THROMBOPENIC PURPURA IN CHILDHOOD. (New England Journal of Medicine, 245:879, Dec. 6, 1951).

On the basis of a study of 47 cases of purpura it was found that children between the ages of 2 and 8 years were most frequently affected. The disease appears to be a benign self-limited condition of short duration. It shows no sex predilection and is rarely familial. A clear-cut relation to antecedent infection or to

allergy could not be demonstrated. There was a suggestive tendency toward a seasonal incidence, the peak of cases occurring in the spring. There were no consistent findings in the bone marrow with regard to either megakaryocytes or degree of eosinophilia. Adequate criteria for splenectomy in idiopathic thrombopenic purpura of children have not been established. Successful results in cases treated by early splenectomy cannot be attributed to the operation, since spontaneous recovery is the rule. Splenectomy in children should probably be reserved for cases of uncontrollable bleeding and chronic recurrent purpura.

MICHAEL A. BRESCIA, M.D.

WATSON, E. M., AND THOMPSON, M. W.: HEREDITY AND DIABETES. (American Journal of Digestive Diseases, 18:326, Nov. 1951).

Although diabetes mellitus shows a marked familial tendency, the exact mode of inheritance of the disease, as yet, has not been determined completely, chiefly because of the absence of full concordance between the diabetic genotype and phenotype. The percentage of cases with positive family histories of diabetes varies inversely with the age at onset of the disease. The age of onset is earlier in those cases with positive family histories of diabetes then in those without such family histories and is especially early in those with bilateral family histories. On the whole, the available evidence favors the hypothesis that diabetes is inherited as a recessive. The age at onset of the disease is positively correlated in sibs but not, or only slightly, correlated in parent-offspring pairs. There is conflicting evidence as to whether diabetes is more likely to appear in the like-sexed than in the unlike-sexed sibs of diabetic propositi, but the present observations suggest that if any such relationship exists, it can be explained on the basis of the preponderance of female diabetics. AUTHORS' SUMMARY.

THE NATIONAL OFFICE OF VITAL STATISTICS OF THE PUBLIC HEALTH SERVICE: LEADING CAUSES OF DEATHS. (Public Health Reports, 67:90, Jan. 1952).

In the first four weeks of life the leading causes of death are immaturity, postnatal asphyxia and atelectasis, birth injuries and congenital malformations. Neonatal deaths account for about two-

thirds of all infant deaths. After the first 4 weeks, until the end of the first year, congenital malformations and pneumonia and influenza are the leading causes. Accidents also first appear as a major cause of death after the first four weeks. In the age group of 1 to 4 years, the main causes of death in order of importance are accidents, influenza and pneumonia, congenital malformations, malignant neoplasms, tuberculosis, gastritis, enteritis and colitis, meningitis, other infective and parasitic diseases, measles and acute poliomyelitis. In the age group of 5 to 14 years, the main causes of death in order of importance are accidents, malignant neoplasms, acute poliomyelitis, influenza and pneumonia, rheumatic fever, congenital malformations, diseases of the heart, tuberculosis, appendicitis and other infective and parasitic diseases. These figures are for the year 1949. MICHAEL A. BRESCIA, M.D.

WALSH, F. M. AND HARRIS, L. D.: OPHTHALMIC HEADACHE. HEADACHE AMONG CHILDREN. (Journal Lancet, 71:530, Dec. 1951).

One hundred children between the ages of 4 to 17 years were examined for refractive errors. Of these, 46 had refractive errors sufficient to cause loss of vision. This group was referred by the school because they failed the vision check. One of these children complained of headache. The remaining 54 children could see well without glasses. This latter group were referred because of symptoms associated with the eyes. The symptoms or reasons for examination of this latter group included, headache, parents want check, eyes hurt, slow reader, holds book too close, general irritability, symptoms with movies, rings under eyes, stumbles and falls, sees dots, line, etc., lids twitch, nausea and speech defect. Children, with refractive errors sufficient to depress their visual acuity, have very few symptoms. However, disturbances of emotional origin are far more significant in the creation of symptoms about the eyes. MICHAEL A. BRESCIA, M.D.

BERGQVIST, S.: ATTEMPTS TO HEIGHTEN THE EFFECT OF BCG VACCINATION WITH HYALURONIDASE. (American Review of Tuberculosis, 64:442, Oct. 1951).

Ninety-five children, approximately 8 years old, were vaccinated with BCG vaccine to which hyaluronidase had been added, and a

control series of 71 children were vaccinated in the usual manner. A tuberculin test (Mantoux, 1 mg. of O.T.) made 29 days later revealed that 61 per cent of those children who had received vaccine together with hyaluronidase were positive reactors, compared with 35 per cent of the control series. The difference, 26 per cent, is statistically significant. Fifty days after vaccination 99 per cent were tuberculin positive in the hyaluronidase group compared to 91 per cent in the control group. The reaction to the vaccination was, in general, stronger among the children who had received the hyaluronidase. An ulceration larger than 10 mm. in diameter developed in 4 of these children. No ulcerations of this size arose in the control group. However, the question of whether the uneven ulceration distribution in the two groups is definitely related to the hyaluronidase administration or perhaps results from some other unknown factor requires further elucidation.

AUTHOR'S SUMMARY.

JACOBSON, G.; COHEN, S. R. AND CARTER, R. A.: PULMONARY COMPLICATIONS OF ACUTE BULBAR POLIOMYELITIS. (Radiology, 57:629, Nov. 1951).

Fifty-three patients with acute bulbar poliomyelitis were studied roentgenologically. Thirty-one (58 per cent) had pulmonary complications, such as pneumonia, atelectasis, pneumatocele, pulmonary over-expansion, pulmonary interstitial emphysema, mediastinal emphysema and pneumothorax. Atelectasis was the most common lesion, occurring in 22 patients, 7 of whom died. Although the lesions were more common in the females (20) as to males (11), 6 of the 7 patients who died were males. An increased amount of intestinal gas was noted in most patients and gastric distention was particularly common. There was no correlation between pulmonary complications and the time of tracheotomy or entry into the respirator after admission, but the time relationship of these latter factors to each other was significant. In 13 out of 17 patients who had tracheotomies one day or more after they were placed in a respirator, pulmonary complications developed. Six of the 7 deaths occurred in this group. No increase in incidence was noted in those who had tracheotomies before or at the time of being placed in a respirator.

MICHAEL A. BRESCIA, M.D.

Major Carl L. Sitter, USMC — Medal of Honor

THE HILL WAS STEEP, snow-covered, 600 feet high. Red-held, it cut our lifeline route from Hagan-ri to the sea; it had to be in our hands.

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Sayer, R. J.; Michel, J.; Mell, F. C., and Kirby, W. M. M.: Am. J. M. Sc. 221:356 (March) 1951

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